0105

Effect and Mechanism of Tamoxifen on Peritoneal Fibrosis in Mice Undergoing Peritoneal Dialysis

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Objective: To investigate the effect and the perhaps mechanism of tamoxifen on peritoneal dialysis related peritoneal fibrosis.
Methods: Intraperitoneal injected with 4.25% glucose peritoneal dialysis fluid to establish a mouse model of peritoneal fibrosis. C57BL/6 male mouse were randomly divided into three groups: control group; high-glucose peritoneal fluid group (PDF group), and tamoxifen group (TAX group). Histologic changes were examined by Masson's trichrome stain. Expression of Wnt4, β-catenin, E-cadherin, NF-κB, COL1A1, cytokeratin protein were evaluated by immunofluorescence staining and Western Blot.
Results: A light microscope Masson's trichrome stain showed the peritonum of mouse in PDF group are significantly thicker than control group; the tamoxifen treatment ameliorated the thickening of peritoneum induced by PDF. Immunofluorescence staining and Western Blot showed that protein expression of Wnt4, β-catenin, NK-κB, COL1A1 increased in PDF group than in control group (all $P < 0.05$), protein expression of E-cadherin and basic cytokeratin decreased in PDF group than in control group (all $P < 0.05$). The tamoxifen treatment ameliorated the increased expression of Wnt4, β-catenin, NK-κB, COL1A1 induced by PDF and the expression of E-cadherin and basic cytokeratin decreased than in PDF group.
Conclusion: Peritoneal dialysis related peritoneal fibrosis may mediated by the activation of Wnt / β-catenin signaling pathway in mouse peritonum, and tamoxifen can ameliorated peritoneal fibrosis possibly by inhibiting the Wnt / β-catenin signaling pathway.

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0106

Efficacy of Low-dose Bisoprolol in Maintenance Hemodialysis Patients with Asymptomatic Left Ventricular Diastolic Dysfunction

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Objective: Left ventricular hypertrophy and diastolic dysfunction are the most frequent cardiac alteration in ESRD. The aim of this study was to determine whether the β-blocker, bisoprolol, has beneficial effects in maintenance hemodialysis patients with asymptomatic left ventricular diastolic dysfunction.
Methods: In this study we enrolled 120 patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis accompanying left ventricular diastolic dysfunction more than six months. Bisoprolol was started with 1.25 mg once daily orally 30 minutes after breakfast and increased every 1 week by 1.25 mg increments up to the maximum tolerated dose. Echocardiographic examination was used to measurement of left ventricular diastolic function.
Results: 108 patients finished 6 months study. After 6 months of treatment, compared with the baseline, the cardiothoracic ratio was significant decreased. Echocardiographic examination showed that there was no significant change in EF, but LVDd, LVDs, PWT were significant decreased. E/A ratio was significantly increased.
Conclusion: Our study demonstrate bisoprolol efficacy in improving left ventricular diastolic dysfunction in HD patient with normal blood pressure.

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0109

Effect of Hemodialysis on Anterior Chamber Biometric Structure and Intraocular Pressure in Non-Diabetic Patients with End-stage Renal Disease

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Objective: To evaluate the short-term changes in the ophthalmologic findings after low-flux hemodialysis in non-diabetic end-stage chronic renal failure (CRF) patients.
Methods: Forty-three patients (86 eyes) selected so as not to have glaucoma or history of glaucoma were studied. We observed the patients on maintenance hemodialysis therapy more than 6 months. Their clinical characteristics and medical records before and after hemodialysis were reviewed through the way of self-control study. Detailed ophthalmologic examinations together with tomometre (intraocular pressure, IOP), A/B scan (lens thickness) and ultrasound biomicroscopy (central anterior chamber depth) were performed immediately before and after HD sessions. Demographic information including age, gender, underling systemic diseases, hemodialysis duration, and levels of body weight, blood urea nitrogen, and creatinine before and after hemodialysis were recorded.
Results: The mean age of the patients at the time of dialysis was 49.7 ± 12.0 (range 33 to 65) years. 53.5% (23) were men. After hemodialysis treatment, the blood urea nitrogen, creatinine, patient weight decreased significantly ($P < 0.01$). There was no significant difference in the change in serum calcium, serum phosphorus, serum albumin and hemoglobin after treatment ($P > 0.05$). Mean central anterior chamber depth also decreased significantly after HD, from 2.46 ± 0.38 to 2.38 ± 0.36 mum (paired t test, $P < 0.01$). Mean lens thickness significantly increased from 4.23 ± 0.22 mm before HD to 4.30 ± 0.12 mm after HD ($P < 0.01$) in group. However, Mean IOP increased from 12.32 ± 4.31 mmHg to 14.31 ± 2.98 mmHg after HD (paired t test, $P < 0.01$).
Conclusion: Conventional hemodialysis can affect the ophthalmologic findings. Patients with chronic renal failure should be checked of their anterior chamber structure and be given corresponding treatment before haemodialysis.

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0110

miRNA-143/145 Gene Cluster Enhances Cell Deformation and Fibrosis of Human Peritoneal Mesothelial Cells via Modulating TPM4

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Objective: This work for the function and mechanism of miR-143/145 gene cluster in human peritoneal mesothelial cells (HPMC).
Methods: To investigate if miR-143/145 gene cluster could promote PM fibrosis, all these immortal and cast-off HPMCs from the effluents of end-stage renal disease (ESRD) patients with peritoneal dialysis (PD) were characterized by fibrosis related markers and tested the expression of miRNA-143/145 cluster and tropomyosin 4 (TPM4) by real time PCR or Western blot. We also used PD dialysis rat model to observe the response of PM to miR-143/145 gene cluster and their possible target.
Results: Here, we found that miR-143/145 gene cluster, which are examined to be highly expressed in HG-induced HPMCs or in cast-off HPMCs ex vivo which have a transformed fibroblastic phenotype stimulated by high glucose (HG, 60 mmol/L) and in PD animal model. TPM4 were found significantly lower expression in HG-induced HPMCs. So our study showed that high glucose from PD fluid could promote the expression of miR-143/145 of HPMCs and reduce the expression of TPM4, compared with normal glucose-cultured HPMCs (Figure 1A-E). Reporter assays further supported that TPM4 were post-transcriptionally regulated together by miR-143/145 gene cluster. Collectively, these data suggested that TPM4 was a downstream target of miR-143/145 gene cluster. Re-expression of miR-143/145 gene cluster by miR-143 or miR-145 mimic led to cell deformation, and reduced cell adhesion, following the down-regulating expression of TPM4 and E-cadherin, but up-regulating expression of α-SMA, CTGF, collagens and fibronectin which might increase PM fibrosis. Depletion of miR-143/145 cluster in HG-induced HPMCs enhanced cell adhesion, marking by up-regulating E-cadherin and TPM4 but down-regulating α-SMA, CTGF, collagens and fibronectin in vitro (Figure 1F).